



August 15, 2008

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U.S. Environmental Protection Agency Region 7  
Mail Code SUPRSPRB  
901 North 5th Street  
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Re: Revised Community Risk Assessment, Herculaneum, Missouri

Dear Mr. Morrison:

Enclosed please find the revised Community Risk Assessment for Herculaneum, Missouri. This report was revised to incorporate the comments received from EPA and MDNR. There are two tables attached to this letter: the first presents a summary of how each comment was addressed, and the second shows how the section numbers changed from the 2006 report to the 2008 report. The remainder of this letter provides additional discussion regarding our response to certain of the Agency's suggested revisions.

EPA Comment 19. EPA requested that the risk assessment use the adult baseline blood lead levels from NHANES III Phases 1 and 2 (1988-1994).

Response: The report was not changed. The report used the NHANES blood lead data from 1999-2000, because these data are more recent than the blood lead data from Phases 1 and 2 of NHANES III (from years 1988-1994), and blood leads have declined since the period 1988-1994. The use of the more recent NHANES data is consistent with the goal of using the most currently available data that reflects the most current science. In other comments, EPA asked that Doe Run use the most currently available data (e.g., the 2007 air lead data), and the most currently available science (e.g. the information on adverse health effects at low blood lead levels from the current NAAQS review). We believe that the 1999-2000 NHANES data published by CDC is valid and appropriate for use in the risk assessment. (See Centers for Disease Control and Prevention (CDC), 2005, "Third National Report on Human Exposure to Environmental Chemicals." July. <http://www.cdc.gov/exposure/report/pdf/thirdreport.pdf>.)

EPA comments 32, 33, 48. EPA requested that the comparison of observed and predicted blood lead levels be removed from the report. MDHSS asked that the report describe the uncertainties in the comparison.

Response: We felt it was important for the report to include the observed blood lead data, thus we retained the comparison of observed and predicted blood lead levels, but included several caveats about this comparison, as suggested by MDHSS (see page 74). As noted in the report, EPA guidance states that comparisons between predicted and observed data are appropriate (US EPA, 1998a; US EPA, 1994b).

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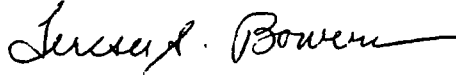
EPA Comment 52. EPA requested that the air modeling to determine the boundary of the exposure area downwind of the slag storage area be revised, to use a newer air model (AERMOD), and local meteorological data.

Response: The air modeling was not updated. The air modeling was performed using the ISCST model, which was replaced by EPA's AERMOD model in 2005. The air modeling was not redone with AERMOD for the 2008 revision of this report, due to the significant expenditure of time and effort that it would have required, without discernable benefit. The only purpose of this modeling was to establish the geographic boundary of the exposure area that is potentially affected by wind-blown slag from the slag storage area. pile. The use of AERMOD with local meteorological data is unlikely to have a substantial impact on the location of this boundary. In addition, a slight adjustment to the location of this boundary will cause a slight change in the number of properties included in Exposure Area 12, but will not impact the calculated risks or the conclusions of the risk assessment. All of the properties in EA 12 are also included in Exposure Areas 1 or 2, and this would not change if the EA 12 boundary were adjusted slightly. In addition, the ISC model was also used for the 2007 SIP modeling.

We plan to finalize the risk assessment report once we receive Agency approval on revisions. We would be happy to schedule a conference call to discuss Agency comments. In the meantime, please feel free to contact us with questions or comments.

Sincerely,

GRADIENT CORPORATION



Teresa S. Bowers, Ph.D.  
Principal

Enclosures

## Responsiveness Summary for Agency Comments on Community Human Health Risk Assessment, Herculaneum, Missouri

EPA Comment	Doe Run Response
<b>General Comments</b>	
1. Additional explanation and detail would greatly improve the overall transparency of the Human Health Risk Assessment (HHRA). The HHRA should be written so as to allow readers to understand all of the steps, logic, key assumptions, limitations, and decisions in the risk assessment. For example, the introduction to several sections should briefly explain the concepts to be discussed in that section to ensure the public can fully understand how the potential health risks have been characterized.	Explanations were added in the introduction to each section.
<b>Specific Comments</b>	
1. <b>Section 1.3 (p. 2)</b> The four parts of a risk assessment discussed in this section should match those outlined in the "Risk Assessment Guidance for Superfund, Part A," (EPA, 1989), as should the general outline of the document (see Exhibit 9-1).	The outline of the report was changed to match the outline in RAGS Part A. As a consequence, all of the section numbers referred to in the agency comments have changed.
2. <b>Section 2.2 (p. 7) (a)</b> This section should also reference and briefly discuss a figure of the conceptual site model that represents the linkages among contaminant sources, release mechanisms, exposure pathways and routes, and receptors. <b>(b)</b> For all residential exposure areas, the risk assessment should assume that child and adult residents live in a single home for 6 and 24 years, respectively, for a total exposure duration of 30 years.	a) CSM figure was added.  b) The text was clarified to state that we used a 24 year exposure duration for adults.
3. <b>Section 3.1 (P. 13)</b> As requested previously by Region 7, the risk assessment must include additional details on soil sample collection (e.g., sieve size, etc). In addition, the discussion should reference Section 3.5 concerning data useability.	Text was added.
4. <b>Section 3.1.2 (p. 15)</b> The regression analysis should be revised to evaluate the correlation between the XRF results as the independent variable "x" and the laboratory results as the dependent variable "y." In addition, all data used in the regression analysis should be provided, as well as the statistical output, including 95% confidence intervals for the regression equation parameters. Doe Run should use these results to determine whether a "correction factor" is warranted to adjust the XRF results to yield a laboratory estimate before calculating an exposure point concentration for lead. This determination should be based on the regression equation for soil concentrations less than 2000 mg/kg because the draft HHRA shows that the correlation varies with concentration and the XRF instrument slightly underestimates laboratory concentrations less than 2000 mg/kg.	We reversed the axes of the regression analysis, included the 95% confidence intervals with the regression, and determined whether a correction to the XRF data was needed.
5. <b>Section 3.2 (p. 16)</b> As requested in previous comments by Region 7, the risk assessment must include additional details on interior dust data sample collection, including sampling methodology, sieve size, presence of lead-based paint, etc.	The available information on dust sampling was added.

EPA Comment	Doe Run Response
<p><b>6. Section 3.2 (p. 17)</b> The risk assessment states that EPA indoor dust data could not be used because property addresses were unavailable. Region 7 will provide these data for inclusion in the risk assessment. Doe Run should also use these data to discuss whether recontamination of home interiors is occurring from the lead smelter.</p>	<p>The properties used for the recontamination study were remediated; therefore, they were not included as properties evaluated in the risk assessment. (No remediated properties were included in this portion of the risk assessment.) We did not receive property addresses from EPA. However, these data are discussed in Section 7.2.5 (Uncertainty Assessment) for an evaluation of the relationship between air lead and dust lead.</p>
<p><b>7. Section 3.3.1 (p. 17)</b> It is unclear why the air monitoring data collected by the Missouri Department of Natural Resources (MDNR) were not included in the risk assessment. These data should be added to the risk assessment and evaluated for potential use.</p>	<p>We added the MDNR air data.</p>
<p><b>8. Section 3.3.1 (p. 18)</b> Doe Run should use the latest air monitoring data which reflect current conditions at the site, as opposed to relying on data collected in 2003.</p>	<p>All properties in the risk assessment were assigned updated air lead data. They were assigned either the 2007 annual average from the nearest air lead monitor, or the modeled value from the 2007 SIP.</p>
<p><b>9. Section 3.4 (p. 22)</b> As requested previously by Region 7, the laboratory detection limits should be provided for each compound listed in Table 5.</p>	<p>The detection limits have been added.</p>
<p><b>10. Section 3.5 (p. 22)</b> While the Risk Assessment Guidance for Superfund (RAGS) Part D Data Useability Worksheets were added to the HHRA, this section must also briefly discuss how the evaluation criteria in EPA's "Guidance on Data Useability in Risk Assessment" have been adequately satisfied for each media.</p>	<p>Text was added.</p>
<p><b>11. Section 5.1 (D. 25)</b> As a point of clarification, ProUCL calculates several estimates of the upper confidence limit (UCL) on the mean concentration and not just the 95% UCL. As previously requested, the risk assessment should provide documentation of the exposure point concentration recommendations generated by ProUCL in a separate appendix.</p>	<p>The ProUCL output sheets for the EPC calculations are provided in an appendix.</p>
<p><b>12. Section 6.3.2 (p. 34)</b> Doe Run should revise the next to last sentence to state "...used to predict BLLs for the child resident, as well as 5 and 6 year old children who attend Taylor School."</p>	<p>The text was changed.</p>
<p><b>13. Section 7.1 (p. 35)</b>            (a) The HHRA should briefly define a reasonable maximum exposure (RME) and central tendency exposure (CTE) scenarios. The text should also indicate that the Herculaneum risk assessment only addresses the RME scenario for non-lead Constituents of Potential Concern (COPCs).            (b) Doe Run should delete footnote 9 because the "Exposure Factors Handbook" (EPA, 1997) indicates that a soil ingestion rate of 200 mg/day is appropriate for RME scenarios. In addition, EPA's "Child-Specific Exposure Factors Handbook" (EPA, 2002) supports the use of 200 mg/day as a conservative mean estimate.</p>	<p>a) The text was changed.             b) The wording in Footnote 9 was changed but not deleted.</p>

EPA Comment	Doe Run Response
<p><b>14. Section 7.1 (p. 36)</b> As previously requested by Region 7, the risk assessment should use a soil adherence factor of 0.2 mg/cm<sup>2</sup> for elementary school children. This value is based on children playing in wet soil and is consistent with RAGS Part E (EPA, 2004) which recommends using a high-end soil contact activity with a central tendency weighted adherence factor for that activity. The text and all tables should be revised accordingly.</p>	<p>The risks were recalculated for the Taylor School using a soil adherence factor of 0.2 mg/cm<sup>2</sup>.</p>
<p><b>15. Section 7.2 (p. 37)</b>  (a) In July 2000, EPA determined that a specific <i>in vitro</i> bioaccessibility assay (IVBA) is considered an appropriate regulatory methodology for estimating the relative bioavailability of lead for quantitative use in site-specific risk assessments (see <a href="http://www.epa.gov/superfund/health/contaminants/bioavailability/transmemo_rel_bio.pdf">http://www.epa.gov/superfund/health/contaminants/bioavailability/transmemo_rel_bio.pdf</a>). The text should be changed to reflect the Agency's new policy, but the risk assessment should continue to rely on the <i>in vivo</i> bioavailability results for predicting blood lead levels.</p> <p>(b) Doe Run has repeatedly told Region 7 that the samples collected for the Casteel <i>et al.</i> (2001) bioavailability study were not representative of the site for unspecified reasons. Region 7 was not present when the samples were collected and was also not notified of the sampling event. The bioavailability report, dated June 2001, is stamped "Draft" and to EPA's knowledge has not been finalized. Thus, Doe Run must acknowledge there are data quality issues associated with this study. As a result, there is significant uncertainty with the study and in comparing the results to more recent bioavailability data.</p>	<p>a) The IVBA results were corrected using the equation on page ES-4 of the EPA, May, 2007 bioavailability document. ("Estimation of Relative Bioavailability of Lead In Soil and Soil-Like Materials Using In Vivo and In Vitro Methods")</p> <p>b) The text about the Casteel <i>et al.</i> (2001) study was modified.</p>
<p><b>16. Section 7.2.3 (P. 40)</b> Doe Run should provide in an appendix containing the statistical output for the various correlation analyses conducted, including 95% confidence intervals for each of the regression equation parameters.</p>	<p>The statistical output is provided in an appendix.</p>
<p><b>17. Section 7.2.3 (p. 42)</b> Given EPA's new policy concerning use of IVBA for predicting site specific bioavailability of lead, it would be appropriate to use the IVBA results for the slag storage pile.</p>	<p>The corrected IVBA results were used for the slag storage pile.</p>
<p><b>18. Section 7.2.4 (p. 43)</b> The equation used to convert IVBA to relative bioavailability (RBA) was revised subsequent to Doe Run's submission of the risk assessment to Region 7. The correct equation is derived in EPA's "Estimation of Relative Bioavailability of Lead in Soil and Soil- Like Materials Using <i>In Vivo</i> and <i>In Vitro</i> Results" (EPA, 2007a) and is listed below:  <u><math>RBA = 0.878(IVBA) - 0.028</math></u>  This equation should be used to estimate RBA values using IVBA results.</p>	<p>The IVBA results were corrected using this equation.</p>

EPA Comment	Doe Run Response
<p><b>19. Section 7.3 (p. 44)</b> Footnote 13 indicates that Region 7 did not respond to Doe Run's submission of alternative baseline blood lead (PbB) and geometric standard deviation (GSD) levels from the National Health and Nutrition Examination Survey (NHANES 1999-2000 and 2001-2002). As a result, Doe Run ultimately chose to use these values in the draft risk assessment. Region 7 did evaluate Doe Run's proposal, but did not formally respond because EPA was conducting its own analysis of the NHANES data, which recently underwent external peer review. Until EPA completes its analysis and evaluates the policy implications of using alternative blood lead values, the risk assessment should use the PbB and GSD values from the Midwest Region in EPA's analysis of Phases 1 and 2 of NHANES III. The alternative values used in the draft risk assessment and their potential impacts on predicted blood lead levels should be addressed as part of the uncertainty discussion.</p>	<p>The report was not changed. The report used the NHANES blood lead data from 1999-2000, because these data are more recent than the blood lead data from Phases 1 and 2 of NHANES III (from years 1988-1994), and blood leads have declined since 1988. The aim of the report was to use the most currently available data.</p>
<p><b>20. Table 14 (p. 47)</b> The Adult Lead Methodology (ALM) should not be used to predict blood lead levels for 8 to 10 year old students attending the Taylor school because it is applicable to women of child-bearing age. Rather, Doe Run should use the Integrated Exposure Uptake Biokinetic (IEUBK) model to predict the blood lead levels of 5 and 6 year old children at the Taylor School.</p>	<p>The IEUBK Model was used for children age 5-7 at the Taylor school. We removed the evaluation of the 8-10 year olds at the Taylor School.</p>
<p><b>21. Section 7.3 (p. 48)</b> The HHRA should clarify how the average inhalation rates for the adolescent trespasser, adolescent recreator, and children at school were derived from the "Exposure Factors Handbook" (EPA, 1997) because it is not readily transparent in the text.</p>	<p>The text was clarified.</p>
<p><b>22. Section 7.4 (p. 49)</b> (a) This section should briefly explain the batch mode for the IEUBK model and why it is used in this risk assessment. In addition, the text should indicate that a child age of 50 months was chosen because the predicted blood lead level for this age approximates the 6- to 84-month average that is calculated in single run mode. (b) The HHRA also should state that the default dietary lead intake estimates were replaced with updated values using food residue data from the U.S. Food and Drug Administration Total Diet Study and food consumption data from NHANES III.</p>	<p>The text was added.</p>
<p><b>23. Section 8 (P. 51)</b> As mentioned in the general comments, additional text should be added to improve the overall transparency of the risk assessment. This section should briefly explain how toxicity assessment is typically performed for both cancer and non-cancer health effects; define toxicity values used in the risk assessment (i.e., reference dose and cancer slope factor); and the process for selecting toxicity values for non-lead COPCs.</p>	<p>The text was added.</p>

EPA Comment	Doe Run Response
<p><b>24. Section 8.2 (p. 51)</b> Doe Run should revise this section to ensure the latest information on the 'potential adverse health effects of lead are discussed by briefly summarizing the conclusions in the "Air Quality Criteria for Lead" (EPA, 2006), which was developed as part of EPA's reevaluation of the existing National Ambient Air Quality Standard (NAAQS) for lead. This lead criteria document (CD) outlines key findings and conclusions regarding adverse health effects, including neurotoxic effects, cardiovascular effects, renal effects, immune system effects, effects on heme synthesis, effects on bones and teeth, reproductive and developmental effects, and effects on other organ systems. The CD concludes that "...Pb effects occur at blood Pb even lower than those previously reported for many endpoints (EPA, 2006)."</p>	<p>The text was added.</p>
<p><b>25: Section 8.2.1 (p. 51)</b> The text calls into question whether neurological effects occur below a blood lead level of 10 µg/dL, when in fact there is overwhelming evidence that neurological effects occur well below 10 µg/dL. The Agency's lead criteria document states "The overall weight of the available evidence provides clear substantiation of neurocognitive decrements being associated in young children with blood-Pb concentrations in the range of 5-10 µg/dL, and possibly somewhat lower (EPA, 2006)." Furthermore, the Agency released its final Staff Paper for the Lead NAAQS on November 1, 2007, which states "In particular, we note that currently available studies provided evidence of adverse health effects associated with blood lead levels and environmental exposures well below those previously identified, and that there is now no discernable threshold for such effects in contrast to the thresholds that had previously been inferred." "As discussed in the CD and summarized in Chapter 3, the current evidence demonstrates the occurrence of a variety of adverse effects, including those on the developing nervous system, associated with blood lead levels extending well below 10 µg/dL to 5 µg/dL and possibly lower." "Further, current evidence does not indicate a threshold for more sensitive health endpoints such adverse effects on the developing nervous system." "In particular, there is now no recognized safe level of Pb in children's blood and studies appear to show adverse effects at mean concurrent blood Pb levels as low as 2 µg/dL (EPA, 2007b)." These conclusions are supported by the Clean Air Scientific Advisory Committee's (CASAC) review of the CD and Staff Paper, which states "Moreover, there is no evidence of a threshold for the adverse consequences of lead exposure; studies show that the decrements in intellectual (cognitive) functions in children are proportionately greater at PbB concentrations &lt; 10 µg/dL..." "There is also compelling evidence that the risks for mortality from stroke and myocardial infarction are increased at PbB concentrations below 10 µg/dL, which is considerably lower than those considered acceptable for adults. Finally, although less definitive, there is also evidence that lead exposure during pregnancy is a risk factor for spontaneous abortion or miscarriage at PbB concentrations &lt; 10 µg/dL." "In fact, this evidence suggests these blood lead concentrations below 5 µg/dL are associated with unacceptable adverse effects (Henderson, 2007)." Last of all, the Centers for Disease Control's Advisory Committee on Childhood Lead Poisoning Prevention recently issued a report stating that "Research conducted since</p>	<p>We added text.</p>

EPA Comment	Doe Run Response
1991 has strengthened the evidence that children's physical and mental development can be affected at BLLs < 10 µg/dL (CDC, 2007)." Doe Run should cite these recent evaluations as well as include key conclusions from the documents which clearly show adverse health effects, including neurological effects, at PbB concentrations below 10 µg/dL.	
<b>26. Section 8.2.4 (p. 52)</b> The discussion of the carcinogenicity of lead is not consistent with EPA's Integrated Risk Information System (IRIS) which classifies lead as a probable human carcinogen. The Staff Paper (EPA, 2007b) also indicates that both the National Toxicology Program and the International Agency for Research on Cancer have concluded that lead and lead compounds are probable human carcinogens. Doe Run should delete the current text citing the American Conference of Government Industrial Hygienists (ACGIH) and replace it with appropriate information from IRIS and the Staff Paper.	The text was updated with information from IRIS and the Staff Paper.
<b>27. Section 8.2.5 (p. 53)</b> As discussed in the comments above, Doe Run should revise this section to ensure the most currently available science is referenced, including the substantial evidence supporting neurological effects in young children with blood lead levels in the range of 5-10 µg/dL and possibly lower.	We added text about EPA's current Lead NAAQS review and acceptable blood lead levels.
<b>28. Section 9.1.2 (P. 55)</b> (a) In comments dated March 3, 2005, Region 7 requested that the cancer risk for children and adults be added together or an age-adjusted approach be used in the HHRA. The cancer risks should assume an exposure duration of 6 years and 24 years for a child and adult, respectively. Doe Run should revise the exposure assessment text and cancer risk estimates accordingly, as well as the derivation of a preliminary remediation goal for arsenic. (b) The word "COC" should be replaced with "COPC" in this section and throughout the document.	a) The cancer risks were revised.  b) The text was revised.
<b>29. Section 9.2.3 (P. 57)</b> This section documents that ingestion of cadmium and arsenic in homegrown produce represents a complete exposure pathway. Thus, the HHRA should quantify the potential health risks from this exposure pathway using the sampling results from the Agency for Toxic Substances and Disease Registry (ATSDR) exposure assessment, if the data are adequate.	The homegrown vegetable intake pathway was not added to the risk assessment because the data in the ATSDR report are not adequate to evaluate this pathway. A comment to this effect was added to the text.
<b>30. Section 10.4 (P. 61)</b> This section should be revised to indicate that only children ages 5 to 7 years old were evaluated at the Taylor School using the IEUBK model (see comment #20).	The text was revised.
<b>31. Section 10.9.1 (p. 65)</b> If possible, the HHRA should summarize the data on blood lead levels for children living in Herculaneum collected by the Missouri Department of Health and Senior Services (MDHSS) for the last 10 years. This summary should include the number of children sampled, minimum PbB, maximum PbB, geometric mean, number and percentage of children greater than 10 µg/dL.	The MDHSS blood lead data were added to the report.

EPA Comment	Doe Run Response
<p>32. Section 10.9.1 (p. 66) Region 7 does not agree that it is standard lead risk assessment practice to compare observed and predicted blood lead levels nor is it appropriate to conduct an empirical comparison on a "broader geographic basis." Empirical comparisons are only appropriate when there is sufficient evidence that the observed blood lead concentrations adequately represent the population and the exposure assumptions in the IEUBK model adequately represent the individual children sampled. In other words, one must ensure that the two populations being compared span similar conditions. It is also important to recall that the IEUBK model is not expected to exactly replicate the observed blood lead concentrations of specific children. Rather, the model is designed to predict the plausible distribution of PbB concentrations for a child or group of children under a given set of exposure conditions. As discussed in EPA (1994) and Hogan <i>et al.</i> (1998), blood lead data should satisfy several criteria before being used as the basis for comparison to IEUBK model blood lead predictions. For example, paired blood lead and environmental lead levels should be collected within approximately 1 month of each other because the IEUBK model assumes exposure concentrations are relatively constant. Environmental lead concentrations must be characterized in all media (soil, indoor dust, drinking water, air, garden produce, etc.) that contribute to a child's exposure to lead. It is also important to collect behavioral and demographic data, including the time spent away from the primary residence and also to ensure that a child has actually lived at the residence for the 3 months preceding the blood lead measurement. If this type of information is not collected, then an empirical comparison is highly uncertain and one would expect there to be differences between predicted and observed blood lead levels. It is evident that these criteria have not been satisfied in the Herculaneum risk assessment and, as a result, no conclusions can be reached by this invalid empirical comparison. Therefore, Doe Run should indicate that the data are not adequate to perform an empirical comparison and delete all remaining text which discusses this issue. Rather, the conclusion of this section should state that the existing blood lead data demonstrate there continues to be a significant health threat from lead in this community and that blood lead levels have declined since 1975. This decline is likely due to a variety of factors, including decreases in airborne smelter emissions, residential yard cleanups, and health education.</p>	<p>The comparison of predicted and observed blood lead levels was retained in the report. However, the caveats suggested by MDHSS were added to the discussion.</p>

EPA Comment	Doe Run Response
<p><b>33. Section 10.9.2 (p. 67)</b> The same general considerations regarding adequate exposure characterization apply to comparing predicted blood lead levels using the Adult Lead Methodology and observed blood lead levels in women of child-bearing age. Once again, the empirical comparison is not valid because Doe Run has inadequate exposure information on the adult resident population and the empirical comparison discussion should be deleted. As with young children, the blood lead data indicate that adolescents and adults have been impacted by lead in the community.</p>	<p>The text was revised.</p>
<p><b>34. Section 11 (p. 69)</b> Risk based concentrations (RBCs) or preliminary clean-up goals (PRGs) should be derived separately from the risk assessment itself. Thus, Doe Run should move this section to a separate appendix.</p>	<p>The RBC calculations are presented only in an appendix. All text about RBCs was removed from the body of the report.</p>
<p><b>35. Section 11.2 (P. 70)</b> (a) The PRGs for arsenic and cadmium should be derived using the same exposure parameters used in calculating risks, which includes accounting for the dermal route of exposure. (b) Per the National Contingency Plan, Doe Run should use the "point of departure" or a cancer risk of <math>1 \times 10^{-6}</math> to derive an arsenic PRG, regardless of whether this value is below naturally-occurring background levels in soil. Region 7 will ultimately determine the appropriate clean-up level when making a risk management decision for the site. Doe Run should revise the arsenic PRG and the text accordingly.</p>	<p>a) We added the dermal route to the RBC calculations.</p> <p>b) The NCP allows cancer risk of <math>1 \times 10^{-6}</math> to <math>1 \times 10^{-4}</math> as acceptable risk level. Therefore we calculated a range of RBCs based on this risk range.</p>
<p><b>36. Section 12.1.1 (p. 72)</b> (a) This section cites Dragun and Chiasson (1991) as providing background surface soil concentrations of arsenic and cadmium in Missouri. However, Region 7 previously informed Doe Run that using background surface soil concentrations that are not site specific values was inadequate and that a statistical hypothesis test should be used to differentiate site-related and background constituents (see "Guidance for Characterizing Background Chemicals in Soil at Superfund Sites" [EPA, 2002]). Because site-specific data are unavailable, Region 7 recommends using the U.S. Geological Survey Pluto Database to characterize the range of background arsenic and cadmium concentrations found in Jefferson County, as well as adjacent counties. If Region 7 determines that remediation is necessary for these two compounds, an appropriate clean-up level will be derived that accounts for naturally-occurring background levels. Doe Run should revise the text accordingly in all sections that reference background levels. (b) Region 7 also does not agree that it is unnecessary to calculate RBCs for arsenic because there are soil concentrations that equate to a Hazard Quotient greater than 1. Doe Run should delete this sentence from the HHRA.</p>	<p>a) The background data from the USGS PLUTO database, and the USGS Geochemical Landscapes database were added to the report.</p> <p>b) All text about RBCs was removed from the body of the report. RBCs were calculated for arsenic.</p>

EPA Comment	Doe Run Response
<p><b>37. Section 12.1.7 (p. 76)</b> In addition to the studies cited in the text, this section should briefly discuss Roberts <i>et al.</i> (2007) which evaluated the relative bioavailability (RBA) of 14 soil samples from 12 different sites. The RBA values range from 5 to 3 1% which provides further support for arsenic bioavailability likely being overestimated in the HHRA.</p>	<p>The text was added.</p>
<p><b>38. Section 12.2.2 (p. 79)</b> The discussion concerning variability of lead concentration as a function of soil particle size should be deleted because Region 7 has recently provided Doe Run site specific data comparing lead concentrations in the fine (&lt; 250 µm) vs. total soil fractions. Doe Run should evaluate and incorporate these data into the risk assessment.</p>	<p>The EPA size fraction data were added to the report.</p>
<p><b>39. Section 12.2.3 (p. 80)</b> In the fourth sentence, the soil ingestion rate should be revised to 100 mg/day, while the fifth sentence should be revised to 200 mg/day.</p>	<p>The text was revised.</p>
<p><b>40. Section 12.2.4 (p. 81)</b> As requested in previous comments by Region 7, the risk assessment should also acknowledge there is additional uncertainty when using <i>in vivo</i> bioavailability estimates for adolescents and adults because evidence exists to indicate that absolute bioavailability of soluble lead (e.g., in food or water) varies with age.</p>	<p>The text was revised.</p>
<p><b>41. Section 12.2.5 (p. 82)</b> Doe Run should provide the output from the regression analysis for the parameters listed in Table 26, including 95% confidence intervals.</p>	<p>The regression analysis output is included in the tables in this section.</p>
<p><b>42. Section 12.2.5 (p. 83)</b> While Figures 17 to 19 seem to suggest that the IEUBK model default equation underestimates indoor dust lead concentrations, the risk assessment must acknowledge that there is significant uncertainty with this analysis because 26 dust samples represents only 3% of the properties, the air concentrations are modeled values, and the presence of other lead sources (e.g., lead-based paint, spillage along haul routes, etc.) is unknown. In addition, there is no statistical analysis to support the conclusion that indoor dust lead concentrations decrease with distance from the smelter (see Figure 19). Thus, Doe Run should revise the last sentence in the second paragraph to state "...that the IEUBK model may underestimate the impact.. .."</p>	<p>The text was revised.</p>
<p><b>43. Section 12.2.5 (p. 84)</b> Doe Run should revise the last sentence to state "...the IEUBK model may underestimate.. ."</p>	<p>The text was revised.</p>

EPA Comment	Doe Run Response
<p><b>44. Section 12.2.5 (p. 84)</b> Region 7 does not agree with the conclusion that "...the focus on soil remediation is misplaced...." Rather, the limited data suggest that reducing airborne lead levels should be the highest priority, but lead found in surface soil also significantly contributes to exposure and elevated blood lead levels. Doe Run should delete this paragraph from the risk assessment and the potential impact on clean-up goals should be addressed in the appendix containing the preliminary remediation goals.</p>	<p>The text was revised.</p>
<p><b>45. Section 12.2.7 (p. 87)</b> Doe Run should delete both paragraphs on this page referring to Appendix H and replace the appendix with the latest version of EPA's "Lead Soil Trend Analysis" prepared by TetraTech EM Inc., dated August 31, 2007. The text in this section should also be revised to reflect EPA's recontamination analysis contained in Appendix H.</p>	<p>The appendix on recontamination was revised, and is now Appendix J. All four of EPA's Lead Soil Trend Analyses are attached in the appendix. A brief summary of the appendix remains in the text.</p>
<p><b>46. Section 12.2.8 (p. 88)</b> Doe Run should delete this section from the risk assessment.</p>	<p>The section on uncertainty in cleanup goals was deleted from the risk assessment.</p>
<p><b>47. Section 13 (p. 90)</b> (a) The summary should also present the percentage of residential properties in each Exposure Area which exceeds EPA's health protection goal. (b) Doe Run should delete all text which discusses risk-based concentrations.</p>	<p>a) We added the percent of residential properties in each Exposure Area that exceed EPA's health protection goal of 400 mg/kg soil lead.</p> <p>b) All text about RBCs was deleted from the main text of the report.</p>
<p><b>48. Section 13 (p. 91)</b> (a) Doe Run should delete the paragraph discussing observed and predicted blood lead levels, per previous comments on this issue. (b) The primary conclusion of this risk assessment is not that it tends to overestimate risks. Rather, Doe Run must revise the third paragraph to state that the environmental data, blood lead data, and predicted blood lead levels clearly demonstrate there is a significant health threat to young children in Herculaneum.</p>	<p>a) The comparison of predicted and observed blood lead levels was retained in the report. However, the caveats suggested by MDNR were added to the discussion.</p> <p>b) This paragraph was revised.</p>
<p><b>49. Section 13 (p. 92)</b> The last two sentences are Doe Run's opinion concerning how soil clean-up levels should be established by EPA. Doe Run should delete these statements which discuss risk management issues and thus, are not appropriate for the risk assessment.</p>	<p>This paragraph was revised.</p>
<p><b>50. Tables 16A and 16B</b> Per comment 28, Doe Run should add another row depicting the total cancer risk for a long-term resident by adding together the adult and child cancer risk estimates.</p>	<p>The tables were revised.</p>
<p><b>51. Figures 9 to 14</b> The term "<i>in vitro</i> bioavailability" should be replaced with "<i>in vitro</i> bioaccessibility" because it is technically inaccurate to indicate that <i>in vitro</i> models measure bioavailability.</p>	<p>The figures were revised.</p>

EPA Comment	Doe Run Response
<p><b>52. Appendix A</b> The text should clarify how these modeling results for air and soil deposition were actually used in the risk assessment. The air modeling provided in Appendix A should be performed using EPA's AERMOD air dispersion model to model emissions from the slag pile. The ISCST model was replaced in November 2005 by EPA's AERMOD model. The AERMOD system includes the preprocessing AERMAP and AERMET models. The AERMOD model has better scientific algorithms and should give more accurate predictions. The meteorological data from the St. Louis International Airport are not representative of the meteorological conditions near the Herculanum smelter. Meteorological data measured from April 1997 to March 1999 at the Herculanum facility should be used in the model instead of the meteorological data from the St. Louis International airport. A more thorough justification for using a model input value of 3.0 meters per second for the threshold wind speed is needed. The calculated threshold wind speeds for the State Implementation Plans (SIPS) ranged from 11 to 19 meters per second.</p>	<p>The air model was not rerun with AERMOD instead of ISCST. Footnote 3 was added to the text to provide further explanation of the air modeling:</p> <p>"The air modeling was performed using the ISCST model, which was replaced by EPA's AERMOD model in 2005. The air modeling was not redone with AERMOD for the 2008 revision of this report, because the only purpose of this modeling was to establish the boundary of the exposure area, and the new model is unlikely to have a substantial impact on this boundary."</p>
<p><b>53. Appendix B</b> The Data Useability Worksheets are missing information in some fields and should be completely filled out so as to fully address each question.</p>	<p>The data useability sheets in Appendix B were filled in with all the information known about the data.</p>
<p><b>54. Appendix D (Tables 2.1 and 2.3)</b> (a) Per previous comments from Region 7, the "Background Value" column should be deleted and the rationale for COPC detection should be revised from "ABV" to "ASL." (b) Doe Run should delete footnotes 3 and 5 which indicate that background values were used to screen COPCs.</p>	<p>a) The tables in Appendix D were revised. This is now Appendix C.</p> <p>b) The footnotes were deleted.</p>
<p><b>55. Appendix D (Table 3.1)</b> The exposure point concentrations for EA 13 and the reference to footnote 4 are missing from this table.</p>	<p>The EPCs for EA 13 were added to the table.</p>
<p><b>56. Appendix D (Tables 4.1 and 4.2)</b> (a) Footnote 3 should be deleted because it is no longer relevant. (b) The reference should be revised to USEPA (2004) in Footnote 4 and in the rest of the document. (c) The grades listed for each school in Footnote 6 should be consistent with the text of the HHRA.</p>	<p>a) The footnote was deleted.</p> <p>b) The reference was revised.</p> <p>c) Footnote 6 was revised.</p>
<p><b>57. Appendix F</b> The tables labeled as "Adolescent Lead Model" should be revised to "Adult Lead Methodology" with the words "Adolescent Receptor" inserted below the first line.</p>	<p>The tables were revised. This is now Appendix I.</p>

**Response to MNDR and MDHSS Comments (3/5/07)  
on Community Human Health Risk Assessment, Herculaneum, Missouri**

<b>MDNR Comment</b>	<b>Gradient Response</b>
1. A discussion on the interaction of metals was not incorporated into the assessment.	A discussion on the interaction of metals was added to the report.
2. Inhalation of fugitive dust pathway was not included. A discussion of this pathway and its potential contribution to site risks should be incorporated.	We added a qualitative discussion of the fugitive dust pathway in Section 3.2.
3. <u>Section 3.3 Air Data Summary</u> states that air samples collected by the U.S. Environmental Protection Agency (EPA) between October 2001 and August 2002 were reported as non-detect for arsenic, cadmium, nickel, and zinc; however, MDHSS believes that this information may be inaccurate and needs to be verified. A health consultation prepared by MDHSS evaluating arsenic and cadmium levels in air and residential soils reports that these compounds were detected in air up to 0.64 µg/m <sup>3</sup> for arsenic and 0.66 µg/m <sup>3</sup> for cadmium.	The text was revised.
4. Section 5.1 Soil EPC - EPCs for lead in soil, dust, and air are incorrectly referenced as being presented in Appendix B.	The text was revised.
5. Section 11.1 Lead – This section incorrectly references Figures 16 and 17 as showing the relationship between a receptor's home soil lead concentration and the RBC for their intermittent exposure area.	The text was revised.
6. Table 11 lists the Exposure Frequency for the Long Term Child Visitor as 52 days/year; however, risks were calculated for 52, 156, and 260 days/year.	The table was revised.
7. Tables 16A to 17B and Appendix E - The tables summarizing cancer risks and non-cancer hazards (Tables 16A to 17B) contain several calculated values that do not correspond to those shown in the Risk Calculation Sheets in Appendix E. Additionally, several calculation sheets for the different receptors are missing from the document.	The tables were revised.
8. Appendix H references a "Table 1" as showing the trend analysis for recontamination data; however, this table appears to be missing from the document.	Appendix H was revised and is now Appendix J.

MDNR Comment	Gradient Response
<p>9. <u>Section 3.3 Air Data Summary</u>  It is noted that MDNR has co-located air monitoring stations and that the MDNR data is not reported in the assessment. No explanation is given as to why the MDNR data is not presented. All available data should be presented and incorporated as applicable in the risk assessment.</p>	<p>The MDNR air data were added to the report.</p>
<p>10. <u>Section 5.2 Soil Lead EPC for Intermittent Exposure Scenarios</u>  The Weighted Soil EPC equation is incorrectly listed. This should be listed as:  Weighted Soil EPC = <math>((3/7) \times (\text{Slag Storage Area Concentration})) + ((4/7) \times (867 \text{ mg/kg}))</math></p>	<p>The text was revised.</p>
<p>11. <u>Section 10.4 Schools</u>  The time-weighted average EPCs listed in the table appear to be incorrect and should be recalculated.</p>	<p>The calculation is correct (185 days at school, 180 days at home). The text was revised to provide additional explanation.</p>
<p>12. <u>Section 10.9 Comparison of Observed and Predicted Blood Lead Levels and Section 13 Summary and Conclusions</u>  These sections incorrectly reference a 2001 blood lead “study” conducted by MDHSS/ATSDR. An actual “study” has not been conducted for Herculaneum, the testing conducted was simply a screening offered to the community as an intervention effort. Therefore, MDHSS recommends that such instances referring to a study be reworded to correctly reference the report as a “health consultation” and the testing conducted as a “screening”.</p>	<p>The text was revised.</p>
<p>13. It is inappropriate to draw conclusions that the IEUBK and ALM models are overpredicting environmental lead risks based on the comparison presented. MDHSS recommends that either observed results simply be presented in the assessment with no comparison made to predicted results or the comparison be revised to include information to qualify the noted differences, and the stated conclusions based on this comparison be stricken from the assessment.</p>	<p>The comparison of observed and predicted blood lead levels was revised by adding caveats to the discussion.</p>

## Changes in Section Numbering from 2006 to 2008 Report

### 2008 report

Section	Title
1	Introduction
1.1	Site Background
1.2	Report Objectives
1.3	Risk Assessment Process
1.4	Report Organization
2	Data Evaluation and Identification of Chemicals of Potential Concern
2.1	Soil
2.1.1	Soil Data Sources
2.1.2	Correlation Between XRF and Laboratory Data for Lead
2.1.3	Soil Remediation
2.2	Interior Dust Data Summary
2.3	Air Data Summary
2.3.1	High-Volume Air Monitoring Data
2.3.2	Pre-SIP and Post-SIP Air Modeling
2.3.3	Comparison of Modeled and Observed Air Lead Data
2.4	Slag Data
2.5	Data Usability Assessment
2.6	Identification of Chemicals of Potential Concern (COPCs)
3	Exposure Assessment
3.1	Exposure Areas
3.2	Exposure Pathways and Receptors
3.2.1	Voluntary Property Purchase Area
3.2.2	Buffer Zone
3.2.3	Residential Areas Outside Voluntary Property Purchase Area
3.2.4	Schools
3.2.5	Residential Areas in Pevely and Crystal City
3.2.6	Residential Area North of Slag Storage Area
3.2.7	Slag Storage Area
3.3	Exposure Point Concentrations
3.3.1	Soil EPC
3.3.2	Soil Lead EPC for Intermittent Exposure Scenarios
3.3.3	Air EPC
3.3.3.1	Air Lead Concentrations in Residential Exposure Areas
3.3.3.2	Air Lead EPCs in Exposure Areas 12 and 13
3.3.4	Dust EPC
3.3.4.1	Dust Lead EPC
3.3.4.2	Non-Lead Dust EPC
3.4	Quantification of Exposure
3.4.1	Ingestion of Soil
3.4.2	Dermal Contact with Soil
3.4.3	Blood Lead Modeling
3.4.3.1	Adult Lead Model
3.4.3.2	IEUBK Model
3.5	Exposure Assumptions
3.5.1	Exposure Parameters for Cancer/Non-Cancer Risks
3.5.2	Bioavailability of Lead
3.5.2.1	In Vivo Studies
3.5.2.2	In Vitro Study
3.5.2.3	Effect of Various Factors on Soil and Dust Lead IVBA
3.5.2.4	Comparison of In Vitro and In Vivo Results
3.5.3	Adult Lead Model Inputs
3.5.4	IEUBK Model Inputs

### 2006 report

Section	Title
1	Introduction
1.1	Site Background
1.2	Report Objectives
1.3	Risk Assessment Process
1.4	Report Organization
3	Data Evaluation
3.1	Soil
3.1.1	Soil Data Sources
3.1.2	Correlation Between XRF and Laboratory Data for Lead
3.1.3	Soil Remediation
3.2	Interior Dust Data Summary
3.3	Air Data Summary
3.3.1	High-Volume Air Monitoring Data
3.3.2	Pre-SIP and Post-SIP Air Modeling
3.3.3	Comparison of Modeled and Observed Air Lead Data
3.4	Slag Data
3.5	Data Usability Assessment
4	Selection of Chemicals of Potential Concern (COPCs)
2	Exposure Scenarios
2.1	Exposure Areas
2.2	Exposure Pathways and Receptors
2.2.1	Voluntary Property Purchase Area
2.2.2	Buffer Zone
2.2.3	Residential Areas Outside Voluntary Property Purchase Area
2.2.4	Schools
2.2.5	Residential Areas in Pevely and Crystal City
2.2.6	Residential Area North of Slag Storage Area
2.2.7	Slag Storage Area
5	Exposure Point Concentrations
5.1	Soil EPC
5.2	Soil Lead EPC for Intermittent Exposure Scenarios
5.3	Air EPC
5.3.1	Air Lead Concentrations in Residential Exposure Areas
5.3.2	Air Lead EPCs in Exposure Areas 12 and 13
5.4	Dust EPC
5.4.1	Dust Lead EPC
5.4.2	Non-Lead Dust EPC
6	Quantification of Exposure
6.1	Ingestion of Soil
6.2	Dermal Contact with Soil
6.3	Blood Lead Modeling
6.3.1	Adult Lead Model
6.3.2	IEUBK Model
7	Exposure Assumptions
7.1	Exposure Parameters for Cancer/Non-Cancer Risks
7.2	Bioavailability of Lead
7.2.1	In Vivo Studies
7.2.2	In Vitro Study
7.2.3	Effect of Various Factors on Soil and Dust Lead IVBA
7.2.4	Comparison of In Vitro and In Vivo Results
7.3	Adult Lead Model Inputs
7.4	IEUBK model Inputs

4	Toxicity Assessment	8	Toxicity Data
4.1	Toxicity Data for Non-Lead COPCs	8.1	Toxicity Data for Non-Lead COCs
4.2	Adverse Effects of Lead Exposure	8.2	Adverse Effects of Lead Exposure
4.2.1	Neurological Effects	8.2.1	Neurological Effects
4.2.2	Effects on Pregnancy and Fetal Development	8.2.2	Effects on Pregnancy and Fetal Development
4.2.3	Effects on Heme Synthesis	8.2.3	Effects on Heme Synthesis
4.2.4	Cancer Effects	8.2.4	Cancer Effects
4.2.5	Current Guidelines for Protecting Children from Lead	8.2.5	Current Guidelines for Protecting Children from Lead
4.3	Possible Synergistic and Antagonistic Interactions		
5	Risk Characterization for Non-Lead COPCs	9	Risk Characterization for Non-Lead COPCs
5.1	Cancer Risks	9.1	Cancer Risks
5.1.1	Risk Calculation	9.1.1	Risk Calculation
5.1.2	Risk Results	9.1.2	Risk Results
5.2	Non-Cancer Risks	9.2	Non-Cancer Risks
5.2.1	Risk Calculation	9.2.1	Risk Calculation
5.2.2	Risk Results	9.2.2	Risk Results
5.2.3	ATSDR Exposure Assessment	9.2.3	ATSDR Exposure Assessment
6	Risk Characterization for Lead	10	Lead Risk Characterization
6.1	Voluntary Property Purchase Area	10.1	Voluntary Property Purchase Area
6.2	Buffer Zone	10.2	Buffer Zone
6.3	Residential Areas Outside of Voluntary Property Purchase Area	10.3	Residential Areas Outside of Voluntary Property Purchase Area
6.4	Schools	10.4	Schools
6.5	Residential Area in Pevely and Crystal City	10.5	Residential Area in Pevely and Crystal City
6.6	Residential Area North of Slag Storage Area	10.6	Residential Area North of Slag Storage Area
6.7	Slag Storage Area	10.7	Slag Storage Area
6.8	Effect of Exposure Frequency on Average Blood Lead Levels for Intermittent Exposure Scenarios	10.8	Effect of Exposure Frequency on Average Blood Lead Levels for Intermittent Exposure Scenarios
6.9	Comparison of Observed and Predicted Blood Lead Levels	10.9	Comparison of Observed and Predicted Blood Lead Levels
6.9.1	Blood Lead Levels for Young Children	10.9.1	Blood Lead Levels for Young Children
6.9.2	Blood Lead Levels for Adults	10.9.2	Blood Lead Levels for Adults
7	Uncertainty Assessment	12	Uncertainty Assessment
7.1	Uncertainty in Non-Lead Risks	12.1	Uncertainty in Non-Lead Risks
7.1.1	Risks Compared to Background	12.1.1	Risks Compared to Background
7.1.2	Uncertainty in Exposure Point Concentrations	12.1.2	Uncertainty in Exposure Point Concentrations
7.1.3	Uncertainty in Human Intake	12.1.3	Uncertainty in Human Intake
7.1.4	Uncertainty in Exposure Frequency and Duration	12.1.4	Uncertainty in Exposure Frequency and Duration
7.1.5	Uncertainty in Dermal Absorption	12.1.5	Uncertainty in Dermal Absorption
7.1.6	Uncertainty in Toxicity Values	12.1.6	Uncertainty in Toxicity Values
7.1.7	Uncertainty in the Bioavailability of Arsenic in Soil	12.1.7	Uncertainty in the Bioavailability of Arsenic in Soil
7.2	Uncertainty in Lead Risks	12.2	Uncertainty in Lead Risks
7.2.1	Uncertainty in Exposure Frequency	12.2.1	Uncertainty in Exposure Frequency
7.2.2	Uncertainty in Soil Exposure Point Concentrations	12.2.2	Uncertainty in Soil Exposure Point Concentrations
7.2.3	Uncertainty in Soil Ingestion Rate	12.2.3	Uncertainty in Soil Ingestion Rate
7.2.4	Uncertainty in Bioavailability of Lead in Soil	12.2.4	Uncertainty in Bioavailability of Lead in Soil
7.2.5	Uncertainty in Estimated Dust Lead Concentrations	12.2.5	Uncertainty in Estimated Dust Lead Concentrations
7.2.6	Uncertainty in Air Exposure Point Concentrations	12.2.6	Uncertainty in Air Exposure Point Concentrations
7.2.7	Uncertainty Associated with Recontamination of Yard Soils	12.2.7	Uncertainty Associated with Recontamination of Yard Soils
		12.2.8	Uncertainty in Risk Based Concentrations
8	Summary and Conclusions	13	Summary and Conclusions
		11	Risk Based Concentrations
		11.1	Lead
		11.2	Non-Lead COCs

Appendix	Title
A	Regression Statistics for XRF and Laboratory Lead Data
B	Data Useability Worksheets
C	RAGS Part D Planning Tables
D	Air Modeling for Slag Storage Area
E	95% UCLM Calculation Sheets for Non-Lead COPCs
F	Data Summary
G	Regression Statistics for IVBA and Various Factors
H	Risk Calculation Sheets for Non-Lead COPCs
I	Risk Calculation Sheets for Lead
J	Analysis of Recontamination Data
K	Risk Based Concentrations

Appendix	Title
A	Air Modeling for Slag Storage Area
B	Data Usability Worksheets
C	Data Summary
D	RAGS Part D Planning Tables
E	Risk Calculation Sheets for Non-Lead COCs
F	Risk Calculation Sheets for Lead
G	RBC Calculations
H	Analysis of Recontamination Data